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**Risk Mitigation during Human  
Electromuscular Incapacitation Research**

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Final Report**

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## ABBREVIATIONS

AED	Automated external defibrillator
CK	Creatine kinase - total
CK-MB	Creatine kinase - cardiac muscle isoenzyme
CK-MM	Creatine kinase - skeletal muscle isoenzyme
CO <sub>2</sub>	Carbon dioxide
dL	tenth (deci) of a liter
ED	Emergency Department
EKG	Electrocardiogram
EMI	Electromuscular incapacitation
HCO <sub>3</sub>	Bicarbonate
HECOE	Human Effects Center of Excellence
HEMI	Human electromuscular incapacitation
Hg	Mercury (from Latin Hydrargyrum)
L	Liter
L2	Second lumbar vertebra
mEq/L	thousandths (milli) of an equivalent per liter
mg	thousandths (milli) of a gram
mL	thousandth (milli) of a liter
mm	thousandths (milli) of a meter
mM/L	thousandths (milli) of a mole per liter
NAMRU-SA	Naval Medical Research Unit-San Antonio
NIJ	National Institute of Justice
O <sub>2</sub>	Oxygen
pCO <sub>2</sub>	Partial pressure of carbon dioxide
PEA	Pulseless electrical activity
pg	trillionth (pico) of a gram
pH	Potential of hydrogen
pO <sub>2</sub>	Partial pressure of oxygen
PVC	Premature ventricular contraction
SEF	Spectral frequency
T6	Sixth thoracic vertebra
TASER©	Thomas A. Swift's electric rifle
TERA	Toxicology Excellence for Risk Assessment

## **EXECUTIVE SUMMARY**

### **Background**

United States military employment of Human Electromuscular Incapacitation (HEMI) began after a 2003 request from US Army forces in Iraq. Currently available HEMI devices operate at multiples of five seconds duration and distances up to 32 feet. Operational forces have requested HEMI capabilities that exceed these current limits. To date only TASER® International, Inc. funded researchers have published studies of extended duration HEMI exposures. To define the risks, the Human Effects Center of Excellence (HECOE) undertook two reviews of risk associated with HEMI. The 2005 review by the Directed Energy Bioeffects Division focused on identifying where effects research was needed, while the 2012 Naval Medical Research Unit-San Antonio (NAMRU-SA) technical report covered HEMI effects research to date. The NAMRU-SA report identified six effects with a risk of significant injury: rhabdomyolysis (muscle injury), cardiac effects, respiratory dysfunction, electrolyte imbalances, seizures, and vertebral compression fractures. These six risks, plus risks identified from civilian law enforcement activities, and HEMI effects research since 2012, are covered in this report.

### **Research Findings**

Muscle injury can occur before symptoms appear. Blood levels of the proteins, creatine kinase (CK) and myoglobin, are used to detect early injury. CK, an enzyme released from damaged muscle cells, increased more than 100-fold for some individuals after a single five second HEMI exposure, though mean CK increases were small. Elevated myoglobin levels pose a risk of kidney damage. No extended exposure HEMI study has measured serum myoglobin levels.

Cardiac effects carry two different risks, rhythm disturbances and myocardial injury. The incidence of cardiac rhythm disturbances is very dependent upon the orientation of HEMI electrodes with respect to the heart. Cardiac capture is the most likely disturbance to occur and was documented during one research study. Ventricular fibrillation has been induced in animals but requires 12 times the energy as required for cardiac capture. Myocardial injury is a special form of muscle injury. Blood biomarkers for this include a specific form of creatine kinase, CK-MB, and Troponin I. Changes can also occur in the electrocardiogram (EKG) with myocardial injury, such as a myocardial infarction or heart attack. No HEMI study has found evidence of myocardial injury, though one case report found EKG changes and elevations of both CK-MB and Troponin I after HEMI use during law enforcement. The electrodes attached close to the thorax overlying the heart, the area with the greatest risk for cardiac rhythm disturbance. No information was given about the HEMI device used.

Respiratory dysfunction may occur due to restricted movement of the muscles of ventilation. Respiratory acidosis is a significant physiological disturbance after EMI stimulation greater than five seconds duration. Results from studies of extended duration EMI exposure in animals show significant respiratory acidosis. Studies of HEMI exposures up to 30 seconds duration do not show this effect, though respiratory data from these studies suggest that participants were hyperventilating prior to the exposures.



Electrolyte imbalances during and after EMI exposure have been shown to cause acidosis, primarily of metabolic rather than respiratory origin. A 10-second HEMI exposure caused significant changes in pH, lactate, and potassium levels.

Seizures are a possibility only if one or both HEMI electrodes attach to the head.

### **Recommendations**

1. Serum CK and myoglobin levels should be monitored for all experimental HEMI exposures.
2. It is prudent to limit extended duration human EMI exposures to 10 seconds until additional data is available to confirm or refute electrolyte level changes reported to date. This also keeps HEMI exposures short until the absence of respiratory dysfunction is verified.
3. Specific monitoring for cardiac muscle injury is not warranted, based upon the currently available literature. Individuals with known cardiac disease or strong risk factors, based upon the American Heart Association/American College of Sports Medicine Health/Fitness Facility Pre-participation Screening Questionnaire should be excluded from participation in HEMI studies.
4. Placing the HEMI electrodes with a right ventral, trans-diaphragmatic orientation appears best to minimize the risk of cardiac rhythm effects and not increase the risk of vertebral compression fracture. This orientation also avoids the risk of a seizure.
5. Pre- and post-exposure skin examination will be required during HEMI experiments to assess the presence or absence of electrical burn. Persons with a history of keloid formation should be excluded from participation in HEMI studies.

## 1.0 INTRODUCTION

This paper will cover six topics included in the 2012 Naval Medical Research Unit-San Antonio (NAMRU-SA) report on Human Electromuscular Incapacitation (HEMI) effects research to date.<sup>1</sup> Cardiac rhythm effects and myocardial injury are presented as separate topics. Additional effects reported in the open literature will be included where relevant to human use research.

HEMI has been in use by law enforcement officers for decades. United States military employment of this technology began after a 2003 request from US Army forces in Iraq. The Joint Non-Lethal Weapons Program funded development of a hand-held device to fulfill this request. Currently available HEMI devices operate at multiples of five seconds duration and distances up to 32 feet. Operational forces have requested HEMI capabilities that exceed these current limits. There is little research on HEMI exposures greater than five seconds and incapacitating a human at distance by a wireless device presents development challenges. To define the risks, the Human Effects Center of Excellence (HECOE) has undertaken two reviews of risk associated with HEMI. The 2005 review by Toxicology Excellence for Risk Assessment (TERA) focused on identifying where effects research was needed, while the 2012 Naval Medical Research Unit-San Antonio (NAMRU-SA) technical report covered HEMI effects research to date.<sup>2,1</sup> Reviews of HEMI device use in civilian law enforcement activities offer additional information on risks from operational exposures.

In 2005, TERA published a technical report, AFRL-BR-TR-2005-0016, titled Human Effectiveness and Risk Characterization of the Electromuscular Incapacitation Device – A Limited Analysis of the TASER®.<sup>2</sup> Five effects were identified as posing major concerns with short duration exposures and two additional effects were associated with extended duration exposures. The short duration effect of interest is electromuscular incapacitation (EMI). The short duration effects of concern included: ocular injury, seizures, ventricular fibrillation, and fall injuries. EMI was found to be reversible with none or minimal medical intervention. Seizures were also predicted to require minimal medical intervention, though there is no experimental data supporting this. Fall injuries were determined to range from minor to life threatening. Ocular injury was dart-related and assessed as requiring medical attention for full recovery otherwise there is a risk of permanent disability. Ventricular fibrillation was cited as life threatening. The extended-duration HEMI effects of potential concern were acute respiratory impairment and rhabdomyolysis. Aspects of exposure relevant to assessment of operational utility were discussed, though they were not felt to directly pertain to research on the human effects of HEMI. These included: dart strike locations, dart penetration locations, distance between dart strikes, effect of clothing, and completion of a circuit. Burns and lacerations were considered to pose minimal severity. This report served as a guidepost for further research efforts.

In 2012, the NAMRU-SA authored a technical report, NAMRU-SA TR 2012-01, titled Risk and Effects Assessment of Electro-Muscular Incapacitation (EMI) Technologies.<sup>1</sup> This was a comprehensive review of published papers relevant to EMI effects. They identified six effect categories with a risk for significant injury. These include: rhabdomyolysis (muscle injury), cardiac effects, respiratory dysfunction, electrolyte imbalances, seizures, and vertebral compression fractures. This report provides a comprehensive review of injury risks associated

with EMI exposures. A review of the pertinent references, relevant papers since 2012, and conclusions from the NAMRU-SA report are included in this current paper to ensure accurate descriptions of EMI effects and risk mitigation.

HEMI devices employed by civilian law enforcement activities primarily consist of devices produced by TASER® International, Inc. Hundreds of studies, reviews, and opinion pieces have been published about TASER® devices. Bozeman et al. published the largest study to date, prospectively collected three years of data on injuries from law enforcement use of conducted electrical weapons.<sup>3</sup> Of a total of 1201 cases, 938 had no injuries, 260 had mild injuries, two had moderate injuries, and one had a severe injury. The majority of the mild injuries were superficial puncture wounds from the electrode darts. Other mild injuries and the two moderate injuries were related to falls. Two mild injuries were superficial burn marks, presumably from the electrical discharges. The severe injury was muscle injury that developed after the targeted individual used crack cocaine, ran from police, struggled, and received three discharges from a TASER®, all on a hot summer day. Two other men died unexpectedly while in police custody of probable medical causes. These findings are consistent with the effects identified in the two HECOE reviews.

Past researchers frequently studied more than one effect in a given study. To better facilitate understanding of a specific EMI risk, relevant portions from a study may be presented in more than one topic area. Research is reported chronologically, to give the reader a view of how EMI effects knowledge has increased over time. In addition to the six topics mentioned, relevant findings from animal and human studies of EMI, such as long duration exposures, are included.

## **2.0 RISKS OF ELECTROMUSCULAR INCAPACITATION**

### **2.1 Muscle Injury**

Muscle injury associated with HEMI may occur due to stressful muscle contraction, like exercising too much, or injury due to the passage of electric current. Because all body tissues are not perfect conductors, some electrical energy is converted to heat, causing local burns. Burns cause the release of cellular contents into the space surrounding cells and these contents eventually enter the circulation. Rhabdomyolysis occurs when the protein myoglobin is released from damaged muscle cells. Myoglobin is injurious to the kidneys. CK is another protein released from injured muscle cells. CK occurs in different forms called isozymes. The CK-MM isozyme is more specific for skeletal muscle, whereas the CK-MB isozyme is more specific for cardiac muscle. CK-MM is a more sensitive marker for skeletal muscle injury than myoglobin. Several studies of HEMI have included CK, CK isozymes, and/or myoglobin to assay for muscle injury.

Ho et al. studied 66 volunteers during a TASER training course in April 2005. Each participant received a five second exposure from a TASER X26 device.<sup>4</sup> Blood samples were collected at baseline, immediately after exposure, at 16 hours postexposure, and at 24 hours post-exposure. Mean serum myoglobin increased from 32.4 ng/mL to 45.5 ng/mL immediately after exposure, and 51.3 ng/mL after 24 hours. These values are within the normal range. The highest

myoglobin level reported was 167 ng/mL for one volunteer immediately after exposure. Mean serum CK levels were 184.1 U/L at baseline and unchanged after exposure, but increased to 221.6 U/L and 242.3 U/L after 16 and 24 hours, respectively. These serum creatine levels show a modest elevation above the normal range and the highest level reported was 909 U/L. Similar changes in myoglobin and CK can be seen with exercise.

Jauchem et al. exposed ten swine to a series of three 5-second EMI bursts, with 5-seconds recovery between bursts.<sup>5</sup> Venous blood samples were taken before EMI exposure, immediately afterward, and then every 30 minutes until 3-hours after the EMI exposure. Serum myoglobin was significantly elevated from baseline at 30 minutes after EMI exposure, though the approximately 5 ng/mL elevation was not clinically significant. Serum myoglobin levels were not reported for the other post-exposure time points. Serum CK-MM and total CK were elevated immediately post-exposure and continued to rise with each subsequent blood sample. All of the elevated CK-MM and total CK levels were of equal or lesser magnitude than levels seen after sustained exercise.

Criscione studied changes in serum myoglobin and total CK levels in 32 volunteers.<sup>6</sup> Volunteers received a five second exposure from a TASER X-26 device. Blood was drawn at baseline, immediately post-exposure, and 24 hours after exposure. Mean serum myoglobin was 36.8 ng/mL at baseline, 44.7 ng/mL after exposure, and 37.9 ng/mL 24 hours later. There was no significant difference at any time point. Mean serum CK was 313.5 UI/L at baseline, 306.1 IU/L after exposure, and 326.5 IU/L 24 hours later. Serum CK was elevated at 24 hours after exposure, though this was not clinically significant. The natural logarithm (ln) of serum CK levels was calculated to normalize the results. The mean ln of serum CK was 5.6 UI/L at baseline, 5.6 IU/L after exposure, and 5.7 IU/L after 24 hours. Again, no significant difference between the three points in time was found.

Strote et al. reviewed 1,101 police reports of EMI discharges and obtained medical records for 866 subjects.<sup>7</sup> Two hundred seventy-one adults were seen in an Emergency Department (ED). Serum CK levels were obtained only for 23 adults. Ten subjects had total CK levels above 1000 IU/L, the upper limit for a normal male adult being 200 IU/L. This represents over 43% of the adults tested, but testing may have been ordered only where there was clinical suspicion of a significant muscle injury. This is a potential limitation of retrospective reviews. Assuming that the ED physicians did not omit anyone with a potential muscle injury, 10 adults out of 271 seen yields a 3.7% incidence of elevated CK. Assuming that the findings from the 23 adults tested can be extrapolated to the untested adults, an additional 108 adults had elevated CK results.

Ho et al. compared serum measures of metabolic activity and catecholamine levels across five different scenarios: a 150-yard sprint plus a 44-inch hurdle, 45 seconds of striking a heavy bag, a 10-second TASER X-25 EMI exposure, sprinting from a trained K-9 unit while wearing a protective suit, and a spray of oleoresin capsaicin spray to the face and neck.<sup>8</sup> Data was analyzed from a total of 60 subjects who were divided into five groups of 12 subjects. Total CK levels were obtained at baseline and 24 hours later. Only the sprint condition showed a significant elevation at the 24-hour point. CK has a serum half-life of 12 hours. Any clinically significant elevation resulting from one of the five different scenarios should still be present 24 hours after cessation of the activity.

Dawes et al. pooled data from five EMI studies.<sup>9</sup> EMI exposure durations ranged from 5 - 30 seconds. Two models of EMI devices and different electrode arrangements were used for each of the experiments. All studies used the TASER X-26 device except the 30-second exposures which utilized the TASER C2 device. Baseline total CK levels ranged from 12 – 1956 IU/L, with a median of 145 IU/L. The change in total CK levels ranged from -1054 to +2309 IU/L, with a median of +26.5 IU/L, for five-second EMI exposures and from -205 to +25452 IU/L, with a median of +303 IU/L, for 10-second exposures. For 30-second exposures total CK ranged from -140 to +364 IU/L, with a median of +47 IU/L. Data was analyzed in aggregate, so a comparison between five-second and 10-second exposures, both which used the TASER X-26, was not made. Both, the 5- and 10-second X-26 exposure studies showed an increase in total CK levels associated with longer EMI exposures, though a statistical analysis cannot be done without the original data.

In a second study, Dawes et al. exposed 53 volunteers to a TASER X3 device for 10 seconds duration.<sup>10</sup> Participants were divided into three groups with different ventral electrode orientation locations: two upper thoracic electrodes, two upper thoracic and one abdominal or thigh electrode, and two upper thoracic and two abdominal or upper thigh electrodes. Each HEMI exposure lasted 10 seconds. Mean serum CK at baseline for Group 1 was 143 U/L, for Group 2 was 126 U/L, and for Group 3 was 183.5 U/L. After 24 hours, mean serum CK for Group 1 was 245 U/L, for Group 2 was 1544.4 U/L, and for Group 3 was 1622.0 U/L. The difference for Group 3 was statistically significant. The authors note that many of the participants were unable to comply with a post-exposure exercise restriction. The use of multiple stimulation electrodes may have contributed to higher CK levels for Groups 2 and 3 through greater muscle activation.

## **2.2 Cardiac Effects - Rhythm Disturbances**

A 2011 report by the National Institute of Justice (NIJ) identified five cardiac rhythm effects as posing a significant risk from HEMI use: cardiac capture, ventricular tachycardia, ventricular fibrillation, atrial fibrillation, and pulseless electrical activity (PEA).<sup>11</sup> Cardiac capture occurs when an external stimulus drives the rate of cardiac contraction. Ventricular tachycardia occurs when the cardiac ventricles are contracting faster than 100 beats per minute, independent of the sino-atrial node rate. This can present as monomorphic or polymorphic activity on an electrocardiogram (EKG). Monomorphic activity indicates a single site within the ventricles is setting the rate of contraction. Polymorphic activity indicates that several sites within the ventricles are firing independent of each other. This is also known as torsades de pointes, which can degenerate into ventricular fibrillation. Ventricular fibrillation exists when contraction of cardiac ventricular muscle fibers occurs in an uncoordinated manner, leading to cessation of cardiac output. Atrial fibrillation exists when cardiac atrial muscle fibers contract in an uncoordinated manner. To date, there have been no instances of atrial fibrillation recorded in association with or as a result of HEMI exposure. PEA occurs when coordinated cardiac electrical activity is present but there is no effective cardiac output. PEA is not a form of cardiac rhythm disturbance and has not been reported as a direct result of EMI exposure. PEA has not been associated with HEMI exposures during law enforcement activities. Of the three cardiac

rhythm effects identifies in the NIJ report, only cardiac capture, ventricular tachycardia, and ventricular fibrillation pose a direct risk for HEMI exposure.

Cardiac capture, or cardiac pacing, is frequently used in clinical settings when the heart's intrinsic pacemaker is malfunctioning. Cardiac capture requires a lower HEMI charge/current than other forms of cardiac rhythm disturbance. Voorhees et al. found that twelve times as much energy is needed to induce ventricular fibrillation as to enable cardiac capture.<sup>12</sup>

Ho et al. recorded EKGs for 32 volunteers during a TASER training course.<sup>Error! Bookmark not defined.</sup> EKGs were obtained at baseline, immediately post-exposure, 16 hours post-exposure, and 24 hours post-exposure. Each EKG was read separately by an independent cardiologist who was blind to experimental conditions. The EKGs for 30 of the volunteers were read as normal for each time point. Two volunteers had consistently abnormal EKGs, one with left ventricular hypertrophy and the other with occasional sinus pause. Actual EKG measurements were not reported.

Nanthakumar et al. found that the orientation of HEMI electrodes determined whether or not cardiac capture occurred with swine.<sup>13</sup> A thoracic orientation, with one electrode in the right parasternal region and the second electrode in the left lateral thoracic margin, was compared to electrodes attached below the inferior costal margin of the right and left anterior hemi-thoraces. The thoracic orientation produced cardiac capture in 79% of EMI exposures versus none with the electrodes placed below the inferior costal margins.

Levine et al. studied 105 law enforcement officers participating in TASER training.<sup>14</sup> Four limb lead electrodes were attached and EKG monitoring done at least five seconds before, during, and five seconds after a five second HEMI exposure. Mean heart rate increased 15 beats per minute, with a range from -10 to +85 beats per minute. The highest heart rate recorded was 190 beats per minute. EKG recordings were deemed inadequate for determining PR interval, QRS duration, and QTc interval. Two rhythm strips were presented showing that both QT shortening and QT lengthening can occur. The authors stated that 12-lead EKGs might allow formal calculation of cardiac conduction intervals.

Valentino et al. studied 11 electrode orientations for TASER X-26 exposures in four swine.<sup>15</sup> Ventral electrodes were used for nine orientations, comparing trans-cardiac (opposite sides of the heart) and trans-diaphragmatic (above and below the diaphragm) orientations (these were not mutually exclusive). Dorsal electrodes were used for two non-trans-cardiac orientations, comparing trans-diaphragmatic and non-trans-diaphragmatic orientations. Each exposure consisted of 10-second discharges with anode and cathode reversal of the electrodes between discharges. Defibrillation was performed as needed. All electrode orientations and anode/cathode pairs were conducted at least twice. Cardiac capture was seen in 31 of 59 exposures (52.5%) with capture in 23 of 27 trans-cardiac orientations. Ventral trans-cardiac electrodes produced cardiac capture for 15 of 15 supra-diaphragmatic and 8 of 12 trans-diaphragmatic orientations. Ventral non-trans-cardiac electrodes produced cardiac capture for 8 of 24 trans-diaphragmatic and 0 of 8 non-trans-diaphragmatic orientations. Cardiac capture was not produced in any of the four dorsal electrode orientations. Ventricular fibrillation was produced in 2 of 9 exposures with a specific cardiac orientation, centered over the point of

maximum impulse. Other cardiac dysrhythmias were seen in 10 of 57 exposures with ventral electrode orientations, though there was no discussion of the specific rhythms seen. Many of these dysrhythmias may simply have consisted of ectopic beats or other benign rhythms.

Beason et al. compared ventricular fibrillation threshold for the X-26 and an in-house built stimulator.<sup>16</sup> Electrodes were placed 15 cm apart in a cardiac orientation. A five second discharge from the X-26 was used as a baseline. None of 10 swine developed ventricular fibrillation. The in-house stimulator was used to determine the energy level that produced ventricular fibrillation for 50% of the exposures. This 50% fibrillation threshold was four to five times greater than the output of the X-26.

Ho et al. produced cardiac capture in one of eight subjects with a new generation HEMI device.<sup>17</sup> Volunteers received a 10-second HEMI exposure from a TASER X3 device. One electrode was over the mid-sternal area while the second electrode was over the right groin. This electrode orientation is approximately 60 degrees from the orientation described by Nanthakumar. Thus, Ho's one subject did not have a thoracic electrode orientation as described by Nanthakumar. One common characteristic of the two studies is that an electrode was placed over or near the mid-sternum. The manufacturer subsequently lowered the device energy output and none of 45 subjects in a second group experienced a cardiac effect.

Dawes et al. exposed 53 volunteers to a TASER X3 device.<sup>Error! Bookmark not defined.</sup> This is a second paper from the preceding study by Ho et al.<sup>Error! Bookmark not defined.</sup> Three different ventral electrode orientation locations were utilized: two upper thoracic electrodes, two upper thoracic and one abdominal or thigh electrode, and two upper thoracic and two abdominal or upper thigh electrodes. This study was designed to look for respiratory function, biochemistry, and catecholamine changes after a 10-second exposure. Cardiac capture occurred in one volunteer. Testing was stopped and TASER International modified the X3 device to lower the output charge. No other episode of cardiac capture occurred with the new device.

If the HEMI device causes cardiac capture and discharges at a rapid rate, it can induce ventricular tachycardia. Sustained ventricular tachycardia after cessation of the HEMI stimulus has not been seen in human research to date. Theoretically, after cessation of a HEMI stimulus, ventricular muscle may continue to automatically discharge even if the cardiac sino-atrial node is firing properly. This condition may occur in a coordinated or uncoordinated manner. If coordinated, then a ventricular rhythm will be seen. If ventricular muscle discharge occurs in an uncoordinated manner, then ventricular fibrillation will result.

Blackwell and Hayllar reported that QTc prolongation occurred after "low voltage," less than 1000 volts, injury in 28 of 212 (14%) cases.<sup>18</sup> QTc prolongation lasted up to six hours after the last exposure. QTc prolongation of 500 msec or greater can spontaneously convert into multifocal ventricular tachycardia, which in turn can degenerate into ventricular fibrillation. This study illustrates that cardiac collapse may not be directly due to HEMI exposure. Other causes for QTc prolongation, including the presence of stimulant or psychoactive drug, may be additive with HEMI and lead to cardiac collapse. Pre-screening of volunteers for medication use that may predispose to adverse cardiac effects, such as stimulants or anti-depressants, is prudent to reduce risk with HEMI exposures.

Wu et al. examined the effect of electrode to heart distance to produce ventricular fibrillation in swine for a specified energy, 5-second EMI exposure.<sup>19</sup> All exposures surgically placed the electrode within the thoracic wall. A location over the mid-point of the right ventricle was chosen. The average distance to initially produce ventricular fibrillation was 17 mm. Shorter distances likely resulted in shunting of the electrical energy through tissue layers above the heart. Subsequent EMI exposure required less separation of electrodes to produce ventricular fibrillation, 13.7 mm. Defibrillation was performed immediately. There was no attempt to record spontaneous recovery of sinus cardiac rhythm. Other regions of the heart may be more or less susceptible for inducing ventricular fibrillation.

Walter et al. studied, in six swine, two 40-second TASER X-26 exposures with a 10-15 second pause in between exposures.<sup>20</sup> A trans-cardiac orientation was used for electrode placement. Cardiac capture, described as ventricular tachycardia/flutter, occurred in all of the animals. One animal developed ventricular fibrillation and died. The other five animals regained sinus rhythm after the exposure.

Lakkireddy et al. compared five different electrode orientations for TASER X-26 exposures in 11 swine.<sup>21</sup> Ventral electrodes were used for three orientations, transthoracic electrodes for one orientation, and dorsal electrodes for one orientation. Five different current levels were tested, including 1X, 3X, 10X, 20X, and 50X the output of a TASER X-26. A standard X-26 was used for the 1X trials and a custom-built unit was used to provide the higher level exposures. Cardiac capture and ventricular fibrillation threshold currents were lowest for trans-cardiac electrodes with one barb over the sternal notch and the other over the point of maximal impulse. (This differs from the statement by Wu that the mid-point of the right ventricle was most sensitive.) Higher threshold currents were required as the electrodes were moved further from the heart, with the dorsal electrode orientation having the highest threshold currents for cardiac capture and ventricular fibrillation.

Swerdlow et al. performed a retrospective review of 56 non-traumatic sudden deaths associated with HEMI exposure.<sup>22</sup> Inclusion criteria required that every person to collapse within 15 minutes of the last HEMI discharge. The presenting cardiac rhythms, from most to least prevalent, were as follows. Twenty-one had no detectable cardiac rhythm, asystole. Asystole is the end result of untreated ventricular fibrillation. Four other cases initially classified as asystole were found to have cardiac rhythms less than five beats per minute. Eleven had pulseless electrical activity. Another had a rhythm for which an Automated External Defibrillator (AED) advised no shock be given, meaning that ventricular fibrillation or ventricular tachycardia was not present. Five (not including the four cases mentioned above) had significant bradycardia with a cardiac rate less than 30 beats per minute. Four had ventricular fibrillation either on an EKG or based upon an AED advice to deliver a shock. The vast majority of these cases, 50 of 56, had stimulants or psychoactive drugs on blood or urine screening. The term "psychoactive drugs" is not further defined by the authors. A major limitation of this review is that the authors only considered cases where ventricular fibrillation was present or presumed, and discounted those cases where asystole was present. Another limitation of this review is that no EKG changes other than rhythm disturbances were reported.



Ho et al. in a third study of electrocardiographic effects, exposed 25 volunteers to a physically exhaustive exercise regimen, verified by a venous pH measurement, and followed this with a continuous 15 second discharge from a TASER X26 device.<sup>23</sup> EKGs were recorded at baseline and immediately after exposure. EKGs were read by a cardiologist blind to experimental condition. All of the EKGs, except one, were interpreted as normal with respect to rhythm or “clinically significant finding.” The one participant’s baseline EKG had monomorphic premature ventricular contractions (PVCs). No PVCs were noted on this participant’s post-exposure EKG. No EKG intervals were presented.

Kroll et al. compared the energy requirements for two different EMI devices to induce ventricular fibrillation in two small swine.<sup>24</sup> All EMI exposures were 15 seconds in duration. Pulses over three times the output of a TASER X-26 were necessary to induce ventricular fibrillation in 3/7 cathode stimulation exposures. Anode stimulation did not cause ventricular fibrillation at any energy level tested. The authors do not mention if ventricular fibrillation was sustained after cessation of the EMI exposure.

Nanthakumar et al. combined EMI exposure with epinephrine infusion to simulate increased sympathetic tone during stress.<sup>Error! Bookmark not defined.</sup> In one of 16 swine exposures, this produced polymorphic ventricular tachycardia that degenerated into ventricular fibrillation. Non-sustained ventricular tachycardia was produced in a second animal.

VanMeenen et al. collected pre- and post-exposure EKGs from 118 law enforcement officers undergoing TASER training.<sup>25</sup> Baseline 12-lead EKGs were obtained the day prior or the morning of exposure. Post-exposure EKGs were obtained between 20 and 22 hours after exposure. No statistically or clinically significant change was found for mean PR interval, QRS interval, QT interval, QTc interval, P-axis deviation, QRS-axis deviation, or T-axis deviation. A major limitation of this study is the long delay between exposure and post-exposure EKGs, which can permit a return to baseline for EKG changes.

Dawes et al. observed 11 volunteers during a 30-second HEMI exposure from a TASER C2 device. Before and after EKGs were compared.<sup>26</sup> All 11 subjects remained in a sinus rhythm. One asymptomatic subject had a non-specific minimal ST depression. Three subjects experienced a heart rate change of more than 10 beats per minute, two had a rate increase and one had a rate decrease. Echocardiography was attempted for six subjects during HEMI stimulation. Three subjects were identified as having a sinus rhythm and the other three had an indeterminate rhythm. No untoward effects occurred as a result of a 30-second HEMI exposure.

Havranek et al. performed continuous EKG and echocardiographic monitoring of 26 volunteers during a 5-second HEMI exposure from a TASER X26 device.<sup>27</sup> Electrodes were attached along the long axis of the heart, from second intercostal space, right mid-clavicular line, to the fifth intercostal space, left anterior axillary line. Mean heart rate increased from a baseline of 89 beats per minute (bpm), to 101 bpm 10 seconds before HEMI exposure, to 130 bpm 10 seconds after HEMI exposure, and returned to baseline by 5 minutes after HEMI exposure. Two subjects showed an unexpected bradycardia 10 seconds after HEMI exposure, with a further reduction by 5 minutes after HEMI exposure. Both subjects had sinus tachycardia at baseline, 106 and 120 bpm. Echocardiography showed one subject’s heart rate as slow as 35 bpm during HEMI

exposure. QTc interval duration increased from a baseline of 410 msec, to 484 msec at 10 seconds after HEMI exposure. A peak QTc interval was recorded as 605 msec, indicating profound QTc prolongation. This QTc interval change did not reach statistical significance. Mean PR interval and QRS duration were unchanged from baseline at any time point.

### **2.3 Cardiac Effects - Myocardial Injury**

Myocardial injury, as with skeletal muscle injury, can occur due to electrical shock. Clinical markers for cardiac muscle injury include CK-MB and Troponin I. CK-MB is an isozyme of CK predominately found in cardiac muscle, versus CK-MM in skeletal muscle and CK-BB in brain tissue. A cardiac insult can cause a detectable release of CK-MB in serum as soon as 2 hours, peaking by 10 to 24 hours, and returning to normal by 48 to 72 hours. Troponin I and Troponin T are more specific for myocardial damage than CK-MB. Troponins can be detected within 3 to 12 hours of cardiac injury, peak at 24 to 48 hours, and return to normal over 5 to 14 days.

Ho et al. studied 66 volunteers during a TASER training course in April 2005.<sup>Error! Bookmark not defined.</sup> Each participant received a 5second exposure from a TASER X26 device. Blood samples were collected at baseline, immediately after exposure, at 16 hours post-exposure, and at 24 hours post-exposure. Troponin I levels were reported as 0 ng/ml at all times. The text of the article mentions that one volunteer had a reading of 0.6 ng/mL after 24 hours, for which this person was hospitalized for further evaluation. No evidence of myocardial infarction was found and the level was <0.3 ng/mL eight hours later.

Jauchem et al. found no significant change in Troponin I or Troponin T in six swine after two sessions of intermittent EMI exposure over 3 minutes, separated by one hour.<sup>28</sup> Jugular venous blood samples were taken before, immediately post-exposure, 30 minutes and one hour after each EMI exposure. Over the course of 2 hours after the first EMI exposure and one hour after the second EMI exposure, there was significant change from baseline for Troponin I and Troponin T levels. After two hours from the first EMI exposure mean serum Troponin I levels were 0.35 ng/mL, with the uppermost value reaching 0.5 ng/mL. Clinically, this is considered an indeterminate level, less than the lower limit for positive diagnosis of myocardial injury.

Vilke et al. studied 32 law enforcement trainees undergoing a 5-second TASER exposure.<sup>29</sup> A 12-lead EKG was performed at baseline and one hour post-exposure. Specific EKG measurements were not reported. Troponin I was assayed at six hours post-exposure. All values were less than 0.2 ng/mL and were considered negative for evidence of cardiac ischemia. The authors concluded there was no evidence of ischemia or interval changes between EKGs.

Sloane et al. measured serum Troponin I levels after a single 5-second HEMI exposure in law enforcement officers participating in TASER X-26 training.<sup>30</sup> At six hours after the HEMI exposure, all serum Troponin I levels were negative ( $\leq 0.2$  ng/mL).

Walter et al. exposed 6 swine to transcardiac EMI for multiple 40-second pulses attempting to look for cardiac capture and ventricular dysrhythmias.<sup>Error! Bookmark not defined.</sup> The investigators measured Troponin I at baseline, after 5, 15, 30, and 60 minutes, after 24 and 48 hours. Mean

Troponin I levels were essentially equal to baseline, except at 24 hours which rose to 0.02 ng/mL. This increase was not statistically or clinically significant.

Lakkireddy et al. measured in 9 swine CK, CK-MB, and Troponin I at baseline, after multiple 5-second exposures, and after conclusion of the EMI exposures.<sup>Error! Bookmark not defined.</sup> No time duration from baseline to the final exposure was mentioned. They reported no significant change in cardiac injury markers. Because several hours must pass before troponin levels may rise, this study provides no insight on the risk of cardiac injury after multiple EMI exposures.

VanMeenen et al. studied 118 law enforcement officers undergoing TASER training.<sup>Error! Bookmark not defined.</sup> Blood samples were obtained the day prior or the morning of exposure. Post-exposure blood samples were obtained a mean of 21.1 hours after exposure. Mean total CK showed no significant differences between pre- and post-exposures levels. Mean CK-MB and Troponin I were reported as zero and not detectable, respectively, for both time points. These results support the absence of myocardial injury after a five second TASER exposure.

Dawes et al. exposed 11 volunteers to a 30-second HEMI exposure from a TASER C2 device.<sup>Error! Bookmark not defined.</sup> Troponin I levels were assayed at baseline, within 2 minutes after cessation of exposure, and 24 hours after exposure. They reported no “positive” troponins at 24 hours post-exposure.

Ho et al. exposed 53 subjects to 10 second HEMI bursts.<sup>Error! Bookmark not defined.</sup> Troponin I levels were assayed at baseline and 24 hours later. None of the pre- or post-exposure Troponin I levels exceeded 0.04 ng/mL, the lowest level considered positive for myocardial injury.

Belen et al. report a case of myocardial infarction in a 37-year old male.<sup>31</sup> Chest pain persisted for 30 seconds after HEMI exposure from an unspecified HEMI device. One electrode dart was in the left sixth intercostal space and one cm left of the mid-clavicular line, while the other dart electrode was near nipple level and two cm right of the (left) anterior axillary line. This placed the electrodes in an approximately horizontal line over the heart. This is close to the orientation that Wu found to produce ventricular fibrillation.<sup>Error! Bookmark not defined.</sup> Elevated ST segment were noted in leads II, III, and AVF, with reciprocal ST depression in leads I and AVL. Cardiac markers peaked 12 hours after the onset of chest pain, Troponin I at 9 ng/mL and CK-MB at 150 U/L. These levels exceeded the normal values. Coronary angiography was normal. No episode of recurrent chest pain, arrhythmia, or heart failure occurred.

## 2.4 Respiratory Dysfunction

Respiratory dysfunction may occur due to inadequate use of ventilatory muscles. The primary ventilatory muscle is the diaphragm. The chest wall and neck muscles are considered accessory muscles of ventilation. Diminished use of these secondary muscles may occur because of restricted motion due to pain or incapacitation as part of the HEMI exposure. Hypoxia is not the greatest hazard of respiratory dysfunction, rather hypercarbia due to inadequate gas exchange in the lungs leads to respiratory acidosis. The normal respiratory rate is about 12 breaths per minute. Thus, HEMI must last longer than five seconds for respiratory acidosis to begin. With longer periods of HEMI exposure, respiratory acidosis can combine with metabolic acidosis, from lactic acid accumulation due to sustained contraction of skeletal muscle. The combination of respiratory and metabolic acidosis can cause significant metabolic changes.

Ho et al. measured serum lactate in 66 volunteers during a TASER training course in April 2005.<sup>Error! Bookmark not defined.</sup> Each participant received a five second exposure from a TASER X26 device. Blood samples were collected at baseline, immediately after exposure, at 16 hours post-exposure, and at 24 hours post-exposure. The mean level at baseline was 15.8 mg/dL, 24.7 mg/dL immediately post-exposure, 18.3 mg/dL after 16 hours, and 19.8 mg/dL after 24 hours. A peak value of 45 mg/dL was recorded immediately post-exposure. Peak levels of 36 and 37 mg/dL were reported for 16 and 24 hours post-exposure, respectively. Levels above 19.8 mg/dL may be considered as elevated.

Jauchem et al. measured blood pH, lactate, pulse oximetry, and partial pressure of carbon dioxide ( $pCO_2$ ) in six anesthetized swine after two sessions of repeated five seconds on and five seconds off EMI exposure over 3 minutes, separated by one hour.<sup>Error! Bookmark not defined.</sup> The mean pH declined from a baseline of 7.4, to 6.9 immediately after the first exposure, and further to 6.85 after the second exposure. Mean blood lactate increased from 1 mM/L at baseline, to > 12 mM/L immediately after the first exposure, and remained above 9 mM/L for the rest of the experiment. Pulse oximetry showed a mean baseline oxygen ( $O_2$ ) saturation of 90%. This declined to a trough of < 60% after the first exposure and a trough of 52% after the second exposure. Of note, the pulse oximeter accuracy was diminished below 85%. Mean blood  $pCO_2$  increased from a baseline of 48 mm Hg, to 100 mmHg after each EMI exposure and returned to baseline by 60 minutes post-exposure. A pH of less than 7 is considered severe acidosis. The elevation of blood lactate and  $pCO_2$  indicates a combined metabolic and respiratory acidosis which accounts for the decrease in pH seen after the first 3 minutes of intermittent EMI exposure. This condition worsened during the second 3 minute intermittent EMI exposure.

Ho et al. reported data for 52 volunteers exposed to 15 seconds of HEMI from a TASER X-26 device.<sup>32</sup> Eighteen volunteers received three 5-second exposures with a one second pause between exposures, and 34 volunteers received a 15-second continuous exposure. Ventilatory measurements included respiratory rate, tidal volume, end-tidal  $CO_2$ , end-tidal  $O_2$ , and minute ventilation. Four phases were assessed: baseline, during HEMI exposure, the first minute after HEMI exposure, and from the second post-exposure minute until a return to baseline. Mean values and 95% confidence intervals were reported. No statistical comparison was reported for ventilatory change from baseline values or for differences between the two exposure groups. Minute ventilation, the product of tidal volume and respiratory rate, reflects the overall metabolic

rate for tissue oxygenation. Both groups in this study showed modest increases in minute ventilation during HEMI, followed by large increases in minute ventilation during the first minute of recovery. End-tidal CO<sub>2</sub> and O<sub>2</sub> showed a modest increase and decrease, respectively, during HEMI exposure, consistent with increased ventilation. These changes were reversed during the first minute of recovery, probably due to respiratory compensation for metabolic change from increased muscle metabolic activity. The small change in minute ventilation during HEMI suggests that ventilatory movements were restricted, and the post-exposure increase in minute ventilation suggests a return of unrestricted ventilatory movements.

Esquivel et al. exposed 10 swine to four sets of five EMI exposures five minutes apart, for a total of 20 exposures.<sup>33</sup> Each exposure was a five second output from a TASER X-26 device. There is no mention of a pause between each five second output from the X-26 device. Thus, each set appears to consist of 25 seconds continuous EMI exposure. Arterial blood samples were drawn after every set of five exposures and hourly until four hours after the last exposure. Assays included pH, pCO<sub>2</sub>, partial pressure of oxygen (pO<sub>2</sub>), blood lactate, and airway pressure. The mean pH decreased from a baseline value of 7.45 to 7.34 during EMI exposure. A corresponding increase in mean pCO<sub>2</sub> level occurred, from 41 mm Hg at baseline to 52.5 mm Hg during EMI, and returned to a normal level two hours after the last exposure. Mean blood lactate increased from a baseline of 1.3 mM/L to 4.0 mM/L during EMI exposure. There was no significant change in pO<sub>2</sub> or airway pressure at any time. These changes indicate a mild acidosis of mixed metabolic and respiratory nature caused by 25 seconds of EMI exposure.

Vilke et al. evaluated 32 law enforcement officers receiving a five second TASER exposure as part of training.<sup>Error! Bookmark not defined.</sup> Minute ventilation, tidal volume, respiratory rate, and end-tidal pCO<sub>2</sub> were assessed at baseline, and post-exposure at one, 10, 30, and 60 minutes. Arterial blood gases and lactate were drawn at baseline, and post-exposure at one, 10, 30, and 60 minutes. Pulse oximetry was assessed at baseline and post-exposure at five, 10, 15, 20, 25, 30, 40, 50, and 60 minutes. Mean minute ventilation, tidal volume, and respiratory rate values were all significantly increased at one minute post-exposure. pH was decreased significantly without clinical relevance at one minute post-exposure. These values returned to baseline levels by 10 minutes post-exposure and remained at baseline thereafter. Lactate levels increased significantly but not to clinically elevated levels at one and 10 minutes post-exposure. pO<sub>2</sub> and pCO<sub>2</sub> did not differ significantly from baseline values at any time. End-tidal pCO<sub>2</sub> did not differ from baseline values after adjusting for multiple comparisons. These small changes indicate a five second HEMI exposure does not cause clinically significant respiratory change.

Walter et al. exposed six anesthetized swine to two 40-second TASER X-26 stimulations with a 10-15 second pause between.<sup>Error! Bookmark not defined.</sup> Central venous blood samples were drawn at baseline, then five, 10, 15, 30, and 60 minutes post-exposure, plus 24 and 48 hours post-exposure. Assays were performed for pH, pCO<sub>2</sub>, bicarbonate, and lactate levels. Venous pH, pCO<sub>2</sub>, bicarbonate, and lactate showed minor changes. Only differences in mean lactate levels at five, 10, and 30 minutes post-exposure reached significant elevation, up to 3.5 mM/L, below the 4 mM/L anaerobic threshold in exercise. The five minute interval between the cessation of EMI exposure and blood collection for lactate assessment may explain why only minor changes were seen.

A second study by Jauchem et al. exposed ten anesthetized swine to a series of three 5-second EMI bursts, with 5-second recovery intervals between bursts. <sup>Error! Bookmark not defined.</sup> Mean blood pH, lactate, SpO<sub>2</sub>, and SpCO<sub>2</sub> were assayed at baseline, immediately post-exposures, and every 30 minutes thereafter until 180 minutes after the initial exposure. The mean pH at baseline was 7.4, which decreased to 7.25 immediately post-exposure, and increased only slightly to 7.3 by 30 minutes post-exposure. By 60 minutes post-exposure mean pH returned to baseline level and remained there. Mean blood lactate levels were 1 mM/L at baseline, increased to 6.0 mM/L immediately post-exposure, and remained there at 30 minutes post-exposure. The mean lactate level declined to 3.0 mM/L at 60 minutes post-exposure. Subsequent lactate assays returned to baseline levels. Mean SpO<sub>2</sub> remained near baseline levels throughout the experiment. These results differ from the findings of Walter et al., where the swine were given the paralyzing agent succinyl choline. The elimination of sustained muscle contraction likely explains this difference.

A second experiment by Ho et al. exposed 38 volunteers to a TASER X-26 device for 15 seconds.<sup>34</sup> Baseline venous blood assays included venous pH, pCO<sub>2</sub>, and lactate. Volunteers completed intense physical exercise to induce anaerobic exhaustion, push-ups followed by running on a treadmill. Then a second blood sample was drawn. This was followed by EMI exposure for 15 seconds while in a supine position. Then a third blood sample was drawn. A final blood sample was drawn 16 to 24 hours after EMI exposure. A significant decrease in mean pH was noted after exercise, from a baseline of 7.38 to 7.23, and remained low at 7.22 after EMI exposure. An increase in pCO<sub>2</sub> was noted after exercise, from a baseline of 46.3 mm Hg to 57.4 mm Hg after exercise which declined to 51.3mm Hg after EMI exposure. Mean lactate increased from a baseline of 1.65 mM/L, to 8.39 mM/L after exercise, and 9.85 mM/L after EMI exposure. All values returned to baseline 16 to 24 hours after the EMI exposure. These results reflect increasing metabolic acidosis from EMI exposure after exercise, with decreasing respiratory acidosis after cessation of exercise. The combination of respiratory and metabolic acidosis result in continued overall acidosis after the EMI exposure.

A third study by Jauchem et al. exposed 10 swine to EMI for 30 or 60 second periods.<sup>35</sup> Venous blood was collected for pH, lactate, pCO<sub>2</sub>, and pO<sub>2</sub> measurements one minute before and after each EMI exposure. Respiratory rate and hemoglobin oxygen saturation were monitored continuously. Results were reported in aggregate, rather than by 30 or 60 second exposure group. Lactate was significantly elevated at all post-exposure times. Venous pCO<sub>2</sub> was significantly elevated, and pH significantly decreased, immediately post-exposure, but both returned to baseline values by 30 seconds after EMI exposure. Venous pO<sub>2</sub> showed no significant change. No substantial breaths were noted during exposures, though the animals seemed to attempt to breathe. Mean respiratory rate decreased from baseline values immediately post-exposure and returned to baseline levels. Hemoglobin oxygen saturation showed no significant change but did increase over time.

A fourth study by Jauchem et al. exposed 10 anesthetized swine to 30 seconds of EMI from a TASER C2 device.<sup>36</sup> Respiration rate and hemoglobin oxygen saturation were continuously monitored by pulse oximetry. Jugular venous blood samples were drawn one minute before and one minute after TASER exposures, then at 10, 20 30, 60, 90, 120, 150, and 190 minutes post-exposure. Blood assays included pH, pCO<sub>2</sub>, pO<sub>2</sub>, and lactate. No effective respiration occurred during EMI exposures, though some animals did breathe during a 0.5 second pause in the C2

device output. Mean respiratory rate declined from a baseline of 46 to 27 breaths per minute immediately post-exposure. Mean hemoglobin oxygen saturation decreased from a baseline of 90% to 68% immediately post-exposure. Mean venous pCO<sub>2</sub> increased from 60 mm Hg at baseline, to 113 mm Hg immediately post-exposure, but was not significantly different from baseline levels by 10 minutes post-exposure. Mean venous pO<sub>2</sub> decreased from a baseline of 45 mm Hg to 31 mm Hg immediately post-exposure. Mean lactate level was 1.6 mM/L at baseline and increased immediately post-exposure, reaching a peak of 14.1 mM/L at 10 minutes post-exposure, and remained significantly elevated through 180 minutes post-exposure. The immediate respiratory acidosis from inhibited respiration during EMI exposure resolved rapidly, while the metabolic acidosis from muscle activity persisted through 180 minutes post-exposure. The combination yielded a significant overall acidosis. Mean venous pH was 7.39 at baseline, dropped to 7.04 immediately post-exposure, and remained significantly depressed through 60 minutes post-exposure.

Dawes et al. studied 11 volunteers exposed for 30 seconds to HEMI from a TASER C2 device.<sup>Error! Bookmark not defined.</sup> Venous blood was drawn at five minutes before EMI exposure, within one minute post-exposure, and 24 hours later. Mean blood pH and lactate were assayed. Continuous monitoring was performed for respiratory rate, tidal volume, pCO<sub>2</sub>, and pO<sub>2</sub>. Mean pH decreased from a baseline of 7.36 to 7.23 immediately post-exposure. Mean lactate levels increased from 1.46 mM/L to 5.63 mM/L immediately post-exposure, indicating increased metabolic acid production. Mean end-tidal pCO<sub>2</sub> decreased from 36 mm Hg at baseline to 27 mm Hg immediately post-exposure, indicating metabolic alkalosis rather than respiratory acidosis occurred. This contrasts with all other studies employing HEMI exposure greater than five seconds duration. Mean end-tidal pO<sub>2</sub> increased slightly during HEMI exposure from a baseline level of 107 mm Hg to 113 mm Hg during exposure. Mean respiratory rate increased from 17 breaths per minute at baseline to 35 during HEMI exposure. Mean tidal volume decreased from 0.93 L at baseline to 0.72 L during HEMI exposure. Mean minute ventilation increased from 15.1 L/min at baseline (about twice normal value) to 20 L/min during HEMI exposure. After HEMI exposure, mean respiratory rate declined to 19 breaths per minute but mean tidal volume and mean minute volume increased to 1.47 L and 25.7 L/min, respectively. These results indicate a reduction in the volume of each breath was more than compensated by an increase in the frequency of breaths during EMI exposure, leading to respiratory alkalosis. This respiratory alkalosis was not sufficient to offset the metabolic acidosis that occurred, hence the drop in pH during EMI exposure. The post-exposure respiratory rate and volumes suggest increased respiratory compensation for continued metabolic acidosis.

A second study by Dawes et al. exposed 53 volunteers to a TASER X3 device for 10 seconds.<sup>Error! Bookmark not defined.</sup> Participants were divided into three groups with different ventral electrode orientation locations: two upper thoracic electrodes, two upper thoracic and one abdominal or thigh electrode, and two upper thoracic and two abdominal or upper thigh electrodes. Venous blood was sampled for pH and lactate at baseline, immediately after HEMI exposure, and every two minutes for 10 minutes. Continuous respiratory data was recorded via a breath-by-breath gas exchange system and reported as aggregate measures. Mean serum pH at baseline was 7.40, immediately post-exposure was 7.36, at two and four minutes post-exposure was 7.35, at six and eight minutes post-exposure was 7.37, and after 10 minutes was 7.39. Mean serum lactate at baseline was 1.32 mM/L, immediately post-exposure was 3.05 mM/L, after two

minutes was 4.52 mM/L, after four minutes was 4.48 mM/L, after six minutes was 4.57 mM/L, after eight minutes was 4.37 mM/L, and after 10 minutes was 4.11 mM/L. The paper includes a table with blood pCO<sub>2</sub>, pO<sub>2</sub>, and HCO<sub>3</sub> results at the same time points as above but the text does not mention whether these were venous or arterial blood results. It must be assumed that these were venous blood results. Mean serum pCO<sub>2</sub> at baseline was 45.1 mm Hg, immediately post-exposure was 50.0 mm Hg, after two minutes was 45.8 mm Hg, after four minutes was 46.4 mm Hg, after six minutes was 44.2 mm Hg, after eight minutes was 43.0 mm Hg, and after 10 minutes was 42.2 mm Hg. Mean serum pO<sub>2</sub> at baseline was 32.5 mm Hg, immediately post-exposure was 28.5 mm Hg, after two minutes was 44.0 mm Hg, after four and six minutes was 45.0 mm Hg, after eight and 10 minutes was 42.0 mm Hg. Mean serum HCO<sub>3</sub> at baseline was 28.0 mEq/L, immediately post-exposure was 28.3 mEq/L, declined to 25.0 mEq/L after six minutes, and rose to 25.8 mEq/L after 10 minutes. These results indicate a mixed metabolic and respiratory acidosis, with incomplete respiratory compensation until 10 minutes post-exposure. At baseline, the mean respiratory rate was 14 breaths per minute, which increased to 15 during HEMI exposure, and 18 during the post-exposure recovery. Tidal volume decreased from a mean baseline value of 1.09 L (normal is 0.5 L), to 1.02 L during HEMI exposure, and increased to 1.28 L during the post-exposure recovery. Mean partial pressure of end-tidal carbon dioxide (PETCO<sub>2</sub>) was 38 mm Hg at baseline, 35 mm Hg during HEMI exposure, and 40 mm Hg during the post-exposure recovery. Mean partial pressure of end-tidal oxygen (PETO<sub>2</sub>) was 107 mm Hg at baseline, 106 mm Hg during HEMI exposure, and 110 mm Hg during the post-exposure recovery. Mean minute ventilation, the product of respiratory rate and tidal volume, at baseline was 16.6 L/min, 17.2 L/min during HEMI exposure, and 22.4 L/min during the post-exposure recovery. This group of subjects showed a baseline tidal volume and minute ventilation about twice that of an average human. The authors offered no explanation for this.

Jauchem et al. performed a fifth study of prolonged EMI exposure in 10 anesthetized swine.<sup>37</sup> A TASER X-26 device was repeatedly cycled on for seven seconds and off for three seconds, for a total duration of three minutes. Thus, each animal received 18 EMI exposures of seven seconds duration each. Respiratory rate and hemoglobin oxygen saturation were continuously monitored with pulse oximetry. Jugular venous blood samples were collected within one minute before and after each EMI exposure. The blood samples were assayed for pH and lactate. Additional venous blood samples were collected at 30, 60, 90, 120, 150, and 180 minutes post-exposure. Respiratory rates were significantly depressed during the EMI exposure. No animal showed respiratory effort during EMI exposure, though breathing did occur during the three second pauses in the surviving animals. Six animals were apneic immediately post-exposure and expired. Survivors remained hypopneic until five minutes post-exposure. Hemoglobin oxygen saturation decreased significantly for all animals, from a mean baseline of about 86%, through the exposure period. Both survivors and non-survivors showed a continued decline of hemoglobin oxygen saturation until four to five minutes after EMI exposure ceased. Survivors reached a nadir of 40% while non-survivors bottomed at about 10%. Mean venous pH was significantly decreased until 90 minutes post-exposure, with a trough of 6.84 for survivors and 6.88 for non-survivors immediately post-exposure. Mean venous lactate increased from a baseline of 1 mM/L to 23 mM/L after EMI exposure and remained significantly elevated from baseline through 150 minutes post-exposure. These results indicate that after three minute EMI exposures respiratory acidosis persisted over 60 minutes. Metabolic alkalosis persisted for at least 150 minutes post-exposure. Overall acidosis was influenced by both respiratory and



metabolic components, with venous pH not reaching near normal levels until 90 minutes post-exposure. The lack of pCO<sub>2</sub> measurement prevents determining whether the metabolic or respiratory acidosis was predominant.

VanMeenen et al. examined respiratory function during five second HEMI exposure.<sup>38</sup> Twenty-three law enforcement officers participating in TASER training volunteered. Blood samples were obtained the day prior or the morning of exposure. Post-exposure blood samples were obtained a mean of 21.1 hours after exposure. Mean serum bicarbonate showed no significant statistical or clinical change between pre- and post-exposure levels. Respiratory flow and breathing gas temperature were recorded, beginning 20 seconds before, during five seconds of exposure to a TASER X26 device, and until 20 seconds after exposure. Tidal volume decreased during inspiration and expiration phases. The change was greater during inspiration than expiration. A graph was provided, but actual values not given. Baseline inspiratory volume and expiratory volumes were shown as roughly 3.2 L and 2.8 L, respectively. During HEMI exposure, inspiratory and expiratory volumes were roughly 0.5 and 1.6 L, respectively. During post-exposure, inspiratory and expiratory volumes were roughly 5.4 and 4.4 L, respectively. Participants with one probe in the mid-scapular region and the other probe below the sacrum showed less impairment of respiratory function. All participants were asked to sniff during HEMI exposure and only 10 recalled tried to do so. During HEMI exposure, two participants exhibited changes in respiratory flow consistent with sniffing. Voluntary breathing efforts possibly were limited due to impairment of remembering the task or restricted respiratory function.

## **2.5 Electrolyte Imbalances**

Electrolyte imbalances and other metabolic changes can occur during HEMI exposures. During acidosis, hydrogen ions move from the blood into cells to be buffered by the numerous proteins within, and potassium ions move from inside cells to the blood. Elevated blood potassium levels, or hyperkalemia, can result. Blood potassium levels above 6.5 mM/L with EKG changes, or levels above 8.0 mM/L regardless of EKG changes, are associated with ventricular arrhythmias, including fibrillation or cardiac arrest.<sup>39</sup> Blood calcium levels also change as blood pH changes, increasing or decreasing by 0.03 mM/L for every 0.1 increase or decrease in pH, respectively. Blood sodium levels are less affected by acidosis. Blood bicarbonate levels decrease when bicarbonate combines with hydrogen ions from acids to form water and carbon dioxide. The carbon dioxide formed is exhaled during ventilation. Blood lactate levels reflect lactic acid produced during anaerobic metabolism in muscle cells. Lactate can influence on blood pH, and elevated levels are eventually converted to bicarbonate.

Ho et al. studied 66 volunteers during a TASER training course in April 2005.<sup>Error! Bookmark not defined.</sup> Each participant received a 5-second exposure from a TASER X26 device. Blood samples were collected at baseline, immediately after exposure, at 16 hours post-exposure, and at 24 hours post-exposure. Mean serum potassium was 4.1 mM/L at baseline, 3.9 mM/L immediately post-exposure, 4.5 mM/L after 16 hours, and 4.2 mM/L after 24 hours. After 16 hours, serum potassium peaked at 5.7 mM/L, a level that warrants clinical observation. After 24 hours, the peak value recorded for serum potassium was 5.2 mM/L which indicated the potassium level was returning to normal. Mean serum bicarbonate was 22.6 mM/L at baseline, 22.0 mM/L

immediately post-exposure, 24.6 mM/L after 16 hours, and 23.8 mM/L after 24 hours. Similarly, peak serum bicarbonate levels showed a slight, clinically insignificant, increase to 29 mM/L after 16 hours, and after 24 hours. Mean serum sodium, chloride, and calcium showed no significant changes from baseline levels for any time after exposure.

Jauchem et al. exposed 10 anesthetized swine to a series of three 5-second EMI bursts, with 5-seconds recovery between bursts.<sup>Error! Bookmark not defined.</sup> Venous blood samples were taken before EMI exposure, immediately afterward, and then every 30-minutes until 3-hours after the EMI exposure. The authors report significant increases in blood sodium, potassium, and calcium immediately after EMI exposure. Mean sodium was 140 mM/L at baseline and rose to 142. Both of these values are within the normal range. Mean potassium was 4.0 mM/L at baseline and rose to 4.7. These values reflect a mid-normal level rising to a high normal level. Mean blood calcium was 1.31 mM/L at baseline and rose to 1.37. The mean baseline value is near the upper limit of normal for humans. The mean immediate post-exposure level is above normal for humans. The authors do not specify whether the calcium values were for total or ionized calcium, though the levels cited are consistent with ionized calcium levels.

A retrospective study by Strote et al. examined the medical records between 1 Jan 2001 and 31 Dec 2006 for 1,101 persons seen within 24 hours of EMI exposures performed by the Seattle Police Department.<sup>Error! Bookmark not defined.</sup> Blood chemistry tests were performed for small subsets of persons evaluated at local hospitals. Ten adults had blood pH assays, two persons had a pH <7.3. Five adults had blood lactate assays and three of these were greater than 2.1 mM/L. Blood potassium levels were assayed for 134 adults and two persons had levels >5.4 mEq/L. Four persons met the definition for “excited delirium.” Blood CO<sub>2</sub> levels were reported as 25, 18, 26, and 10 mEq/L. These are actually HCO<sub>3</sub> levels, and for compensated metabolic acidosis/alkalosis, correspond to pCO<sub>2</sub> levels of 41, 33, 42, and 23 mm Hg. This summary of heterogeneous medical records provides information only sufficient to state that a minority of HEMI exposures are associated with acidotic changes.

A second study by Ho et al. divided 60 volunteers into five groups of 12 participants with between group comparisons in tasks that simulated different arrest conditions.<sup>Error! Bookmark not defined.</sup> Group 1 completed a 150 meter sprint followed by a 44-inch wall hurdle. Group 2 completed 45 seconds of striking a heavy bag (punching, kicking, etc.). Group 3 underwent a 10-second TASER X26 exposure. Group 4 sprinted 49 meters, then presented a protected arm for a 20-second K-9 engagement. Group 5 received a 2-3 second exposure to oleoresin capsaicin (Pepper Spray) on the face and neck. Peripheral venous blood samples were taken at baseline, immediately after the exposure, then every two minutes, until 10 minutes after the exposure. Baseline mean blood pH ranged from 7.32 to 7.37 for all groups. The sprint and heavy bag conditions caused the greatest decrease in immediate post-exposure pH, 7.16 and 7.04, respectively. Smaller decreases in pH were seen for the TASER and K-9 exposure groups, 7.29 and 7.26, respectively. There was no change in pH for the Pepper Spray group. At eight minutes post-exposure, the TASER exposure group had returned to baseline mean pH level. At 10 minutes post-exposure, the K-9 group was near mean baseline pH level, 7.31. The mean pH levels for the Sprint and Heavy Bag conditions remained decreased, with the Sprint group showing a modest increase toward the baseline mean pH level, but the Heavy Bag group mean pH level remaining decreased at 7.06. Similarly, the mean blood lactate levels showed the

greatest increase immediately post-exposure for the Sprint and Heavy Bag groups, with continued elevated levels through 10 minutes post-exposure. Again, the TASER and K-9 groups showed intermediate increases in mean blood lactate level, though these remained elevated at 10 minutes post-exposure. The Pepper Spray group had no significant change in mean blood lactate level at any time. Baseline mean blood potassium showed no difference between groups, with an overall level of 4.0 mEq/L. The Sprint and TASER groups decreased immediately post-exposure, by 0.2 mEq/L and 0.3 mEq/L, respectively, and remained decreased through 10 minutes post-exposure. The Heavy Bag group had an immediate post-exposure decrease of 0.4 mEq/L, returned to baseline by eight minutes post-exposure, and increased by 0.3mEq/L at 10 minutes post-exposure. No results were reported for the Pepper Spray group. The increase in lactate level reflects a metabolic acidosis, likely due to skeletal muscle anaerobic metabolism. The decrease in pH is influenced by this lacticemia, though other factors may contribute to the overall acidosis. The decline in serum potassium for Groups 1-4 suggest that blood samples were taken during recovery, not immediately post-exertion.

A third study by Ho et al. exposed 38 volunteers to HEMI from a TASER X-26 device for 15 seconds.<sup>Error! Bookmark not defined.</sup> Volunteers completed intense physical exercise to induce anaerobic exhaustion, push-ups followed by running on a treadmill. Then a second blood sample was drawn. This was followed by HEMI exposure for 15 seconds while in a supine position. Then a third blood sample was drawn. A final blood sample was drawn 16 to 24 hours after HEMI exposure. Mean blood potassium was 3.9 at baseline. The authors did not state what units were used, though mM/L is equivalent to mg/dL. The mean serum potassium level rose to 4.2 after exercise, decreased to 3.8 after EMI exposure, and reached 4.1 the next day. The increase in blood potassium immediately after exercise may reflect a shift from intracellular to extracellular spaces or damage to muscle cells such that intracellular potassium was released. The discussion of this study under the Respiratory Dysfunction section explains how a mixed respiratory and metabolic acidosis occurred. All of these potassium levels are considered within the normal range for humans.

In a second study by Jauchem et al. 10 anesthetized swine were exposed to 30 or 60 seconds of EMI from a device similar to a TASER X-26 and the animals were monitored until 180 seconds after exposure.<sup>Error! Bookmark not defined.</sup> Mean blood glucose was not elevated after a single five second EMI exposure, but was modestly elevated between 105 and 135 mg/dL for the 30 and 60 second exposures. Mean blood sodium and potassium levels were increased immediately after EMI exposure, to 144 mM/L and 6.2 mM/L respectively, but returned to baseline levels by the next assay time after end of EMI exposure.

Lu et al. exposed 15 swine to EMI in a two phase study.<sup>40</sup> Phase 1 exposed swine to an initial 30 seconds of EMI, then a five second pause, followed by alternating five seconds on and five seconds off until death. Phase 2 exposed swine to either 12 or 22.5 minutes of EMI exposure. Venous blood samples were collected every five minutes during EMI, immediately post-exposure, and every 15 minutes after EMI termination for an additional four hours. Results from the Phase 2 exposures are presented numerically in Tables 1 and 2.

Table 1. Blood chemistry changes during 12 minutes of electromuscular incapacitation exposures.

Blood Test	Baseline	5 min	10 min	12 min	1 hour	2 hours	3 hours	4 hours
pO <sub>2</sub> (mm Hg)	78	27	26	36	46	46	54	56
pCO <sub>2</sub> (mm Hg)	72	>115	112	98	76	72	80	72
pH	7.32	6.98	6.89	6.91	7.05	7.19	7.23	7.29
Lactate (mg/dL)	1	>15	>15	>15	>15	10.2	6.4	5.2
Sodium (mM/L)	137	144	144	147	140	137	136	135
Potassium (mM/L)	3.8	7.6	7	6.6	4.2	4.7	5	5
Glucose (mg/dL)	65	135	205	200	170	120	105	100
Calcium (mM/L)	1.39	1.44	1.39	1.36	1.29*	1.28*	1.31*	1.29*

\* Survivors (N = 6)

Table 2. Blood chemistry changes during 22.5 minutes of electromuscular incapacitation.

Blood Test	Baseline	5 min	10 min	15 min	20 min	22.5 min	1 hour	2 hours	3 hours	4 hours
pO <sub>2</sub> (mm Hg)	82	24	34	32	36	46	47	50	49	50
pCO <sub>2</sub> (mm Hg)	74	>115	106	110	108	90	84	72	68	64
pH	7.34	6.92	6.88	6.87	6.86	6.9	6.98	7.16	7.27	7.28
Lactate (mg/dL)	1	>15	>15	>15	>15	>15	>15	12	9	7.2
Sodium (mM/L)	140	148	148	148	148	147	143	143	139	139
Potassium (mM/L)	3.9	9.2	6.8	6.8	6.3	6.2	4.7	5.2	5.3	5.2
Glucose (mg/dL)	75	160	195	215	220	235	220	170	130	115
Calcium (mM/L)	1.38	1.44	1.36	1.36	1.36	1.32	1.22	1.24	1.30	1.30

Mean blood sodium increased by five minutes of EMI, and remained elevated until one hour after EMI. The normal upper limit of sodium for swine is approximately the same as for humans, 145 mM/L. This result shows a slight rise in blood sodium that is not of clinical consequence. Mean blood potassium peaked after five minutes of EMI, and decreased by 10 minutes of EMI. The human upper limit of normal for potassium is about 4.9 mM/L. This level shows a significant elevation, with a risk for ventricular arrhythmias. Mean blood calcium was 1.38 mM/L at baseline, increased to 1.44 mM/L after five minutes, declined to 1.35 mM/L after 10 minutes and remained there until cessation of EMI. Mean blood calcium decreased to a nadir of 1.21 mM/L at 90 minutes after cessation of EMI and gradually increased over time, plateauing around a level of 1.29 mM/L for times greater than two hours after cessation of EMI. Beyond five minutes of EMI exposure, the modest decline in blood calcium level is consistent with the level of acidosis observed. Mean blood glucose peaked at 205 mg/dL prior to cessation of EMI in the 12 minutes exposure group but peaked at 235 mg/dL in the 22.5 minutes exposure group when cessation was terminated. Mean blood glucose declined to the upper limit of normal for both exposure groups by four hours after cessation of EMI. The persistent elevation of pH, with elevated lactate levels and normal carbon dioxide levels despite depressed levels of oxygen, indicates that metabolic acidosis was the primary cause of acidosis during prolonged EMI exposure.

VanMeenen et al. studied 118 law enforcement officers undergoing TASER training.<sup>Error! Bookmark not defined.</sup> Blood samples were obtained the day prior or the morning of exposure. Post-exposure

blood samples were obtained a mean of 21.1 hours after exposure. Mean serum sodium, potassium, chloride, calcium, and glucose showed no significant differences between pre- and post-exposures levels.

Dawes et al. exposed 53 volunteers to a TASER X3 device for 10 seconds.<sup>Error! Bookmark not defined.</sup> Venous blood was sampled for electrolytes at baseline and immediately after HEMI exposure. Results were reported in aggregate rather than by exposure group. Mean baseline sodium, potassium, ionized calcium, and glucose showed mild increases after HEMI exposure, but the changes were not of clinical significance.

## 2.6 Seizures

Three case reports for one of a pair of HEMI electrodes attaching to the head were found in the medical literature. Bui et al. report that a generalized tonic-clonic seizure with residual neurological symptoms ensued from the HEMI exposure.<sup>41</sup> Chandler described an individual with a normal neurological examination an indeterminate time after a five second HEMI exposure.<sup>42</sup> Rehman, et al., reported a period of unconsciousness “for almost 5 minutes” after a HEMI exposure, apparently of 5 seconds duration.<sup>43</sup> This person had a normal neurological examination with a mild headache and feeling “shaken” as the only symptoms.

Lu et al. exposed 15 swine to three different electrical stimuli via cranial electrodes.<sup>44</sup> The three different waveforms included: a typical electroconvulsive therapy (ECT) stimulus used as a positive control, a waveform (“EMI”) similar to a commercially marketed EMI device, and an in-house developed waveform (“HEMI”). The latter two waveform exposures were of three different durations presented sequentially: 60, 120, or 180 msec; separated by at least a five minute pause or until a seizure was induced. The “EMI” waveform induced a generalized tonic-clonic seizure in one of 10 exposures lasting 60 seconds, and an absence (petit mal) seizure in one of nine exposures lasting 120 seconds, but no seizure in nine exposures lasting 180 seconds. Of note, the “HEMI” waveform failed to produce a seizure for any duration. The authors relate this to the 95<sup>th</sup> percentile spectral frequency (SEF) of total spectral power. Significant increases in SEF were found only for the “EMI” waveform.

## 2.7 Vertebral Compression Fractures

There are two repeatedly publicized cases of law enforcement officers suffering vertebral compression fractures during training exposures with the TASER X-26 device.<sup>45,46</sup> TASER modified training materials to include a six part informed consent document that mentions the possibility of fractures occurring as a result of TASER exposure. There are only three reports of TASER associated fractures in the medical literature.

Winslow et al. reported that an otherwise healthy 38-year-old man suffered compression fractures of the T6 and T8 vertebrae, plus anterior wedging of the L2 vertebra, as the result of a five second HEMI exposure during law enforcement training with the TASER X-26.<sup>47</sup>

Sloane et al. reported that a 37-year-old man suffered a compression fracture of the T7 vertebra as the result of a five second HEMI exposure during law enforcement training with the TASER X-26.<sup>48</sup> The radiologist commented that this individual had diffuse osteopenia greater than expected for his age. Osteopenia is a loss of bone mineral density, though not as severe as osteoporosis. It can place a person at increased risk factor for fracture. The authors recommended obtaining spinal radiographs for any individual complaining of back pain after HEMI exposure. This strategy will not reduce the risk of fracture.

Vilke et al. reported that one law enforcement officer participating in a study of HEMI physiological effects, suffered a thoracic compression fracture during a five second HEMI exposure with a TASER X-26 device.<sup>49</sup> TASER darts were fired into each subject's back between the shoulder blades from a range of two to three meters. No other details were provided. The authors modified the study procedures such that all subjects were lying on one side upon a mat with alligator clips attached to the left upper anterior thorax and the right waistline. The intention was to decrease the force of contracture of the strong back muscles. No data was provided to show any difference in the strength of muscle contraction between the original electrode positions and the new positions.

## 2.8 Other Findings

### 2.8.1 Long Duration Exposures

Dawes et al. exposed 53 volunteers to a TASER X3 device for 10 seconds.<sup>Error! Bookmark not defined.</sup> Venous blood was assayed for the catecholamines: epinephrine, norepinephrine, and dopamine. Sampling was done at baseline, immediately after HEMI exposure, and every two minutes for 10 minutes. Mean serum epinephrine at baseline was 55 pg/mL, immediately post-exposure increased to 402 pg/mL, at two minutes post-exposure declined to 132.5 pg/mL, and continued to decline until returning to baseline level at eight minutes post-exposure. Mean serum norepinephrine at baseline was 295.5 pg/mL, immediately post-exposure increased to 546.5 pg/mL, at two minutes post-exposure declined to 400 pg/mL, and remained elevated through 10 minutes post-exposure at 373.5 pg/mL. Mean serum dopamine at baseline was 13.5 pg/mL, immediately post-exposure increased to 18.5 pg/mL, and remained elevated through 10 minutes post-exposure at 18 pg/mL. Blood pressure and heart rate declined from baseline levels. Mean systolic blood pressure at baseline was 151 mm Hg, immediately post-exposure was 145 mm Hg, and

10 minutes post-exposure was 138 mm Hg. Mean diastolic blood pressure at baseline was 85 mm Hg, immediately post-exposure was 83 mm Hg, and 10 minutes post-exposure was 77 mm Hg. Pulse differential, the difference between systolic and diastolic blood pressure, at baseline was 66 mm Hg, immediately post-exposure was 62 mm Hg, and 10 minutes post-exposure was 61 mm Hg. Mean heart rate at baseline was 95 beats per minute, immediately post-exposure was 93 beats per minutes, and 10 minutes post-exposure was 88 beats per minute. This may represent a baseline increase of blood pressure and heart rate in anticipation of HEMI exposure or that the increase in catecholamines was not sufficient to affect post-exposure blood pressure and heart rate.

Werner et al. exposed swine to a first EMI exposure of 60 seconds duration, then 60 minutes of rest under anesthesia, followed by a second EMI exposure of 180 seconds duration.<sup>51</sup> A total of 18 swine were exposed with six animals each being exposed to the TASER X26, TASER C2, and Stinger S-200 device. Three animals died during the experiment, one animal died four minutes after the second EMI exposure, and two animals died five and six minutes after the first EMI exposure. Pulse oximetry, hemoglobin oxygen saturation, end-tidal CO<sub>2</sub>, and EKG were monitored during the experiment. Arterial blood samples were collected at baseline, immediately after the first EMI exposure, just before the second EMI exposure, halfway through the second EMI exposure, three minutes after the second EMI exposure, and five minutes after the second EMI exposure. The authors chose to present the data as differences between the three exposure groups and survival, rather than as a comparison to baseline values. Serum cortisol, catecholamines, lactate, and arterial blood gases were assayed. Mean serum cortisol levels showed no significant difference over time for any group. Mean serum epinephrine and norepinephrine levels increased after the first EMI exposure, with the highest level being seen for the two animals that expired after the first EMI exposure. For the remaining animals, mean serum epinephrine and norepinephrine levels rose during the second EMI exposure and declined after completion of the exposure. Mean serum dopamine levels did not change after the first EMI exposure for the two animals that died. For the remaining animals mean serum dopamine levels increased to a plateau after the first EMI exposure, then increased during the second EMI exposure, and declined after completion of the exposure. Mean serum lactate levels rose after the first EMI exposure and continued to rise through the second EMI exposure, though the Stinger S-200 animals had a decline in lactate between the first and second EMI exposures. The arterial blood gases results were reported only as not statistically different between survivors and non-survivors. All animals showed a mixed metabolic and respiratory acidosis. The authors reported the three animals that died had degenerative cardiomyopathy with low vitamin and selenium levels. They conclude that because all of the animals were obtained from the same source and on the same diet that the low Vitamin E and selenium levels were likely not relevant. No other explanation for the degenerative cardiomyopathy was proposed. Pulseless electrical activity developed prior to death, with no finding of ventricular tachycardia or ventricular fibrillation. The authors postulate that this was due to a sudden decline in blood pressure after EMI exposure ended.

Jenkins et al. evaluated the effects of up to 30 minutes continuous EMI exposure in 10 swine.<sup>52</sup> Four animals died during EMI exposure, two after about four minutes of exposure and two more after about 10 minutes of exposure. The surviving animals received EMI exposures of 10, 20, 25, 30, 30, and 30 minute's duration. All animals developed a mixed metabolic and respiratory acidosis. The authors chose to emphasize the data as differences between deceased and surviving animals, rather than as comparisons to baseline values. Mean serum lactate increased within one minute of EMI exposure and continued to increase until reaching a plateau of 19 mM/L at 15 minutes. Mean serum pH, base excess, and bicarbonate all declined upon EMI exposure and continued decreasing until reaching a plateau at 15 minutes, with values of 6.9, 18mM/L, and 16 mM/L, respectively. During a pilot study, the animals were dying due to hypoventilation with subsequent hypoxia. For this study, all animals were ventilated using a constant pressure mode. Ventilation tidal volume decreased during EMI due to muscle stiffness. After 60 to 90 seconds diaphragmatic breathing was observed. Mean statistics were presented for mean pCO<sub>2</sub> and pO<sub>2</sub> values across time. For survivors, mean pCO<sub>2</sub> increased from a baseline of 36.85 mm Hg, to 109.08 mm Hg by three minutes, after which pCO<sub>2</sub> reached a plateau. In contrast, for non-survivors mean pCO<sub>2</sub> increased to 81.43 mm Hg by three minutes and then declined to 72.0 mm Hg by four minutes. For survivors, mean pO<sub>2</sub> decreased from a baseline of 439.33 mm Hg, to 323.6 mm Hg by two minutes and varied about this level afterward. For non-survivors, mean pO<sub>2</sub> decreased to 192.75 mm Hg by two minutes, increased to 233.0 mm Hg by three minutes, and varied about this level thereafter. Mean serum lactate continually increased for survivors, from a baseline of 1.27 mM/L, to 18.79 mM/L by five minutes. For non-survivors, mean serum lactate increased from a baseline of 1.23 mM/L, to 10. 25 mM/L by two minutes, and increased slightly after this to 12.25 mm Hg by five minutes. Statistical significance was seen for the differences in mean serum pCO<sub>2</sub> and lactate, but not pO<sub>2</sub>. A larger sample population may have detected a statistically significant difference for pO<sub>2</sub>. These results show an association between higher measures of serum acidosis and survival. No obvious explanation was found. It was observed that after an initial period of anaerobic exercise, the muscles relaxed and entered a temporary aerobic condition. This could explain the rise in lactate, with buffering causing a decrease in bicarbonate, rise in pCO<sub>2</sub>, and decrease in base excess.

### **2.8.2 Skin Changes**

None of the EMI research literature specifically discusses the effect on skin. The 2005 TERA technical report, AFRL-BR-TR-2005-0016, found burns and lacerations had minor operational significance.<sup>Error! Bookmark not defined.</sup> Some articles on law enforcement use of HEMI mention the risk of laceration and infection associated with removing the barbs deployed by current TASER devices. Many TASER training courses use alligator clips to attach the electrodes to the skin, which avoids laceration and infection.



### **2.8.3 Electrode Placement**

Ho et al. studied the effect of electrode location and spread on ability to complete a goal-directed task during HEMI.<sup>53</sup> Thirty law enforcement officers were exposed for up to 20 seconds while attempting to cross a 10-foot distance and touch a target with a rubber knife. Electrode orientation were varied by right or left side, front or back, and spread (4, 6, 9, 12, 16, and 20 inches). The authors concluded that a minimum electrode spread of 9 inches is necessary to confidently prevent the goal-directed movement toward a target.

## **3.0 DISCUSSION**

### **3.1 Muscle Injury Risks**

Evidence from the majority of six studies shows muscle injury occurring in association with HEMI. None of the laboratory-based studies found CK levels exceeding that seen during exercise. Jauchem noted muscle injury in swine after repeated five second EMI exposures, though none of the findings were greater than seen during physical exercise. Criscione found no clinically significant changes in serum CK or myoglobin after five second HEMI exposures in humans. A retrospective review by Strote of 271 ED visits after TASER exposure showed CK elevations >1000 IU/L in 10 of 23 patients. Only 8.5% of patients had CK assays done and no explanation of other potential causes for CK elevation was given. Ho found no elevation of CK 24-hours post-TASER exposure. A meta-analysis of five studies by Dawes found mean increases in CK of 26.5, 303, and 47 IU/L after 24 hours, for TASER exposures of 5, 10, and 30 seconds duration, respectively. Individual CK elevations increased more than 25,000 IU/L after a single 5-second TASER exposure. Though mean CK increases were small, individual changes were clinically significant.

Serum myoglobin levels were elevated in two five-second HEMI exposure studies. These increases were not clinically significant. Only one swine study with extended duration EMI exposures has measured serum myoglobin levels. Serum myoglobin levels were elevated but not of clinical significance. Because of the potential for kidney damage, it is prudent with extended duration HEMI exposures to measure serum myoglobin levels to verify that no clinically significant increase occurs.

Serum CK and myoglobin levels should be monitored for all experimental HEMI exposures.

### **3.2 Cardiac Rhythm Disturbance Risks**

Voorhees reported that the ventricular fibrillation threshold for mongrel dogs is 12 times the cardiac capture threshold. Nanthakumar found in swine that an electrode orientation over the long axis of the heart had a greater likelihood of producing cardiac capture than a trans-abdominal orientation. Valentino studied electrode orientation in four swine and produced cardiac capture for 23 of 27 ventral trans-cardiac electrode trials, zero of eight ventral non-trans-diaphragmatic electrode trials, and zero of four dorsal electrode trials. Beason reported that the

50% ventricular fibrillation threshold for 10 swine was four-to-five times the output of a TASER X-26.

Cardiac rhythm effects have been documented when HEMI electrodes are attached to the anterior thorax, particularly when aligned with the cardiac long axis. Placing HEMI electrodes on the posterior thorax will reduce, if not eliminate, the risk of cardiac effects. One drawback of this approach is that it is not suitable for any evaluation of cardiac effects. Evaluations of HEMI cardiac effects will require the use of an electrical load target or an animal model before considering human studies to avoid inducing serious change in cardiac activity. A second drawback is that posterior thoracic electrode placement increases the risk of a vertebral fracture. (This topic is discussed later in this paper.) A right ventral, trans-diaphragmatic orientation of electrodes appears best to minimize the risk of cardiac rhythm effects and not increase the risk of vertebral fracture. Alternative electrode orientations can be used when knowledge about specific effects is sought.

### **3.3 Myocardial Injury Risks**

None of the above studies found evidence of myocardial injury with EMI exposures up to 3 minutes. One case report of myocardial infarction was associated with HEMI exposure involved dart electrodes over the heart. As for preventing cardiac rhythm effects, a right ventral, trans-diaphragmatic orientation of electrodes will minimize the risk of myocardial injury.

### **3.4 Respiratory Dysfunction Risks**

Respiratory acidosis is a significant physiological disturbance after EMI stimulation greater than five seconds duration. Esquivel and Walter exposed anesthetized swine to continuous EMI of 25 and 40 seconds duration, respectively. Both found mild respiratory and metabolic acidosis that resolved by 60 minutes post-exposure. Jauchem exposed anesthetized swine to three minute cyclic sessions of EMI exposure in two different studies, the major difference being five seconds on and five seconds off versus seven seconds on and three seconds off. The seven second EMI exposure bursts resulted in a greater reduction in respiratory rate and a 60% lethality rate. Collaborators Ho and Dawes performed human EMI exposure studies of 10, 15, and 30 seconds' duration. The shorter duration exposures resulted in an expected increase in  $p\text{CO}_2$  while the 30 second exposure showed a decrease in  $p\text{CO}_2$ . Restricted ventilatory movement can occur during HEMI, with a resultant decrease in tidal volume and minute ventilation. Compensation can occur through an increased respiratory rate, as evidenced by a mean of 35 breaths per minute in the first study by Dawes. Lung volumes reported in the two studies by Dawes are consistent with hyperventilation from volunteers actively taking deep breaths. Verbal communication indicates that Ho and Dawes had volunteers actively coached to breath during EMI exposures. Pain might also cause an increase in respiratory rate. Neither pain response, nor a voluntary increase in respiratory effort, is possible with appropriately anesthetized swine.

Tidal volume includes gas movement through the trachea and bronchi, where no gas exchange takes place, approximately 150 mL, known as anatomic dead space. The decrease in tidal volume during HEMI exposure yields a greater percentage decrease in effective gas exchange than the simple decrease in tidal volume indicates. With an average tidal volume of 0.5 L, an

average anatomic dead space of 0.15 L results in an effective gas exchange of 0.35 L or 70% of the tidal volume. A decrease in tidal volume of only 10% to 0.45 L, with the same anatomic dead space of 0.15 L, yields an effective gas exchange of 0.30 L, a 15% reduction, not 10%. Any percentage increase in respiratory rate must be greater than the percentage decrease in tidal volume to maintain the baseline effective gas exchange.

Hypercarbia, the buildup of carbon dioxide, gives the initial drive to breathe during inadequate ventilation. Hyperventilation to the point of respiratory alkalosis, noted by subjective paresthesias, can minimize the possibility of hypercarbia. Hypoxia, the lack of adequate oxygen, gives the second drive to breathe during inadequate ventilation. By combining hyperventilation with breathing 100% oxygen for one minute to prevent hypoxia, individuals have been able to stay submerged in water for over twelve minutes without breathing. Respiratory alkalosis and hyperoxia are not suitable when assessing metabolic effects, but may be useful during extended HEMI exposures when assessment of muscle incapacitation is the primary outcome measure. It is prudent to limit human EMI exposures to no more than 15 seconds duration until the results of Ho and Dawes are verified.

### **3.5 Electrolyte Imbalances Risks**

Electrolyte imbalances during and after EMI exposure have been shown to occur with acidosis, primarily of metabolic rather than respiratory origin. The magnitude of acidosis increases with longer durations of EMI exposure, up to a certain point, and frequently with  $\text{pH} < 7.20$ , defined as severe acidosis. A 10-second HEMI exposure caused significant changes in pH, lactate, and potassium levels. It is prudent to limit extended duration human EMI exposures to 10 seconds until additional data is available to confirm or refute electrolyte level changes reported to date.

### **3.6 Seizure Risks**

Seizures are a possibility if one or both HEMI electrodes attach to the head. If cranial attachment is a study condition, then the actual waveform utilized appears to influence the possibility of seizure activity. By deliberately placing the HEMI electrodes upon the torso, the risk of a seizure is eliminated.

### **3.7 Skin Changes**

Anecdotal comments from a USAF Security Forces TASER instructor indicates that skin trauma, due to minor electrical burns, may occur with the use alligator clips. No report of this was found in the medical literature. Observation will be needed to determine the presence or absence of electrical burn. This should be included in the Informed Consent Document as a possible risk of participation in a HEMI experiment. Standard medical treatment with topical corticosteroid preparations can be used to minimize tissue reaction should an electrical burn be identified. Intralesional injection with a corticosteroid can be used if topical preparations are not effective. Individuals with a history of keloid formation are at increased risk for permanent scarring and should be excluded from HEMI exposures.

#### **4.0 RECOMMENDATIONS**

Serum CK and myoglobin levels should be monitored for skeletal muscle injury with all experimental HEMI exposures.

It is prudent to limit extended duration human EMI exposures to 10 seconds until additional data is available to confirm or refute electrolyte level changes reported to date. This also keeps human EMI exposures below 15 seconds duration until the absence of respiratory dysfunction is verified.

Placing the HEMI electrodes with a right ventral, trans-diaphragmatic orientation appears best to minimize the risk of cardiac rhythm effects and not increase the risk of vertebral compression fracture. This orientation also avoids the risk of a seizure.

Specific monitoring for cardiac muscle injury is not warranted, based upon the currently available literature. Since blood testing will be necessary for muscle injury and electrolyte imbalance assessments, adding cardiac enzymes will not increase human subject risk and may benefit overall HEMI physiology knowledge as longer duration exposures are proposed. Individuals with known cardiac disease or strong risk factors, based upon the American Heart Association/American College of Sports Medicine Health/Fitness Facility Pre-participation Screening Questionnaire should be excluded from participation in HEMI studies.<sup>54</sup>

Pre- and post-exposure skin examination will be required during HEMI experiments to assess the presence or absence of electrical burn. This should be included in the ICD as a possible risk of participation in a HEMI experiment. Persons with a history of keloid formation should be excluded from participation in HEMI studies.

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